

# Alcohol and sleep apnea

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*Acute ingestion of ethanol induces vasodilation and swelling of respiratory mucosa; it depresses respiratory centers resulting in hypotonia of oropharyngeal dilator muscles and inducing or aggravating sleep apnea. However, no association between the sleep apnea syndrome (SAS) and Alcohol Use Disorders (AUD) has been demonstrated.*

## Introduction

Sixty eight patients diagnosed as having SAS by polysomnography over a 6-month period were evaluated for the presence of AUD using the Michigan Alcoholism Screening Test (MAST) and an alcohol consumption history questionnaire. The prevalence of AUD was obtained by including both the positive MASTs and/or with admitted hazardous consumption known to correlate with significant physiologic risk (5 or more standard drinks per day on a regular basis). The correlation between moderate and severe SAS known to be associated with increased risk for mortality (Apnea Index > 20), was then evaluated with respect to presence or absence of AUD.

The prevalence of AUD in the SAS patients as defined by MAST alone was 22% (15/68), a figure which correlates with the prevalence found in other studies of in- and outpatient

cohorts. Twelve sleep apnea patients (18%) admitted that they consumed hazardous amounts of alcohol. The prevalence of AUD, using a positive MAST and/or admitted hazardous consumption, was 31% (21/68). Eighty six percent (18/21) of the patients with AUD were found to have an apnea index of 20 or greater, indicating a high mortality risk. AUD and high mortality risk sleep apnea were in significant correlation ( $N = 68$ ,  $r = 0.24$ ,  $p < 0.05$ ).

Our study demonstrates a significant correlation between AUD and SAS, even in the absence of acute alcohol ingestion immediately prior to the polysomnographic test. In addition, the correlation between AUD and SAS severity suggests that the morbidity seen in both AUD and SAS may be related to the same pathophysiologic mechanism.

## Background

The effect of alcohol on sleep is well known. Acute alcohol ingestion disrupts the normal sleep pattern by reducing sleep latency and increasing wakefulness in the last half of the night<sup>1</sup>. Abnormal sleep patterns occur in alcoholics, even after prolonged sobriety, because of the effects of chronic alcohol toxicity on sleep centers<sup>2,3</sup>.

The relationship between acute alcohol ingestion and sleep apnea has also been investigated. Taasan et al demonstrated that 2 ml/kg of alcohol consumed 1 hour prior to bedtime induced apneic episodes (cessation of breathing at the nose and mouth for 10 seconds or more<sup>4</sup>) in normal subjects<sup>5</sup>. Scrima and colleagues found similar results in both normal subjects, as well as in individuals diagnosed with the SAS after 3 oz of 80-proof alcohol<sup>6</sup>. Alcohol induces apnea through both central and peripheral mechanisms. Acute ingestion of ethanol induces vasodilation and swelling of respiratory mucosa and depresses respiratory centers, resulting in hypotonia of oropharyngeal dilator muscles and inducing or aggravating sleep apnea<sup>7,8</sup>. Peripherally, alcohol causes vasodilation resulting in pharyngeal edema<sup>7</sup>.

There is also a possible link between chronic heavy alcohol ingestion and sleep apnea. Vitiello and colleagues demonstrated a high prevalence of nocturnal hypoxic episodes in a population of recovering alcoholics<sup>9</sup>. In addition, the severity of the alcohol abuse was found to correlate with the level of nocturnal hypoxia<sup>9,10</sup>. It was suggested that the mechanism was sleep

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apnea, however these patients were not tested by polysomnography.

The relationship between chronic alcohol use and the SAS (5 or more apneic episodes per hour of sleep<sup>4</sup>) is important because both diseases result in significant morbidity. In fact, the sequelae of both diseases include hypertension, arrhythmias, depressive symptoms, cardiac disease, cerebrovascular accidents and, most importantly, death. He et al demonstrated that sleep apnea patients with an apnea index (AI) > 20 had significantly greater mortality than those with an AI < 20<sup>11</sup>. The correlation between SAS severity and the mortality in alcoholic SAS patients has not been studied.

Our study investigates 2 aspects: (1) What is the prevalence of AUD in patients diagnosed with SAS by polysomnography? and (2) is AUD related to the severity of SAS? The term "Alcohol Use Disorder" (AUD) was chosen to conform with the criteria for alcohol abuse and/or alcohol dependence found in the third edition of the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM III-R)<sup>12</sup>.

### Methods

Sixty eight patients presenting to the Kuakini Medical Center Sleep Laboratory for polysomnography over a 6-month period were diagnosed as having SAS. These patients were evaluated for a possible associated AUD, using the Michigan Alcoholism Screening Test (MAST). MAST is a weighted, 25-item questionnaire, which screens for the presence of alcoholism when 5 or more points are calculated on the basis of positive responses<sup>13</sup>.

The results of MAST were then correlated with the alcohol consumption history obtained from the sleep laboratory questionnaire developed by Laughton Miles<sup>14</sup>. The alcohol consumption questions solicit information concerning the type of alcoholic beverage consumed and the pattern of use as measured by both quantity and time frame. Individuals with admitted consumption of 5 or more standard drinks on a regular basis (daily or on weekends) were considered to have a possible AUD because Sanchez-Craig and colleagues demonstrated that this pattern of "hazardous consumption" results in a significant morbid risk to individuals<sup>15</sup>.

Finally, the relationship between the severity of AUD and of SAS was examined by comparing MAST results and the presence or absence of hazardous consumption of alcohol. The severity of SAS was defined by an apnea index of < 20 versus ≥ 20. An apnea index of 20 is thought to require treatment because of the increased risk of death as a consequence. Such patients are generally considered to be candidates for treatment with continuous positive airway pressure because it has been demonstrated that the incidence of mortality is reduced<sup>11</sup>.

### Results

The average age of patients referred to the sleep laboratory was 48 years, with a range of 20 to 73 years. The prevalence of MAST positive sleep apnea patients was 22% (15/68). None of the 12 female patients were screened positive on MAST, whereas 15 of 56 males were MAST positive for a prevalence of 27%. Fourteen individuals evaluated by the sleep laboratory did not complete MAST; the participation

**TABLE 1**

**Relationship between admitted hazardous consumption and MAST scores**

		POS. MAST	NEG. MAST	TOTAL
<b>HAZARDOUS CONSUMPTION*</b> (Number of Patients)	<b>Present</b>	6	6	12
	<b>Absent</b>	9	47	56

\* Hazardous consumption is the admitted consumption of 5 or more standard drinks on a regular (daily or on weekends) basis.

**TABLE 2**

**Relationship between alcohol use disorders and sleep apnea syndrome**

	SEVERITY OF SAS		AGE	
	Apnea Index <20 (No. of Pts.)	>20 (No. of Pts.)	Total No.	Mean (SD**)
<b>Present</b>	3	18	21	46.8 (7.7)
<b>AUD*</b>				
<b>Absent</b>	18	29	47	48.5 (12.8)

\* Patients with Alcohol Use Disorders (AUD) are MAST positive and/or admit to Hazardous Consumption.

\*\*Standard Deviation

rate was 85%, therefore.

Twelve sleep apnea patients (18%) admitted to regular hazardous consumption of alcohol. Six of the 15 MAST-positive patients did not complete the alcohol consumption questionnaire. The correlation between MAST scores and reports of hazardous consumption was significant (N = 68, r = .27, p < 0.05), as shown in Table 1.

Since our study addressed the impact of both chronic alcohol abuse and dependence on sleep apnea, the number of SAS patients with positive MASTs and/or hazardous consumption were combined. This produced a combined prevalence of 31% (21/68). The combined prevalence in males was even higher 38% (21/56); none of the females screened positive on MAST or indicated hazardous consumption.

Using an Apnea Index (AI) of 20 as a cut-off score to evaluate risk for mortality, 86% (18/21) of the patients with AUD were found to have a high mortality risk related to their sleep apnea (Table 2). AUD and high mortality risk sleep apnea were significantly correlated (N = 68, r = 0.24, p < 0.05).

### Discussion

The prevalence of AUD in the SAS population as determined by MAST was 22%, a figure which compares with the

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prevalence found in other studies of in- and outpatient medical populations<sup>16,17,18</sup>. However, MAST did not identify 9% (6/68) of patients with admitted hazardous consumption. The definition of AUD in the DSM III-R includes individuals who knowingly consume alcohol in the face of medical problems exacerbated by the consumption<sup>2</sup>. In the sleep laboratory, SAS patients with hazardous consumption would fit this definition. Although there was a significant correlation between MAST and the presence or absence of hazardous consumption, the 9% false negative rate was greater than in a study by Cyr and Wartman, who found that in an ambulatory practice population only 7 of 242 individuals were heavy drinkers who did not screen positive on MAST<sup>19</sup>.

MAST might not identify certain hazardous drinkers because the weighted items on MAST generally screen for social consequences of alcoholism, which not all alcoholics develop. The medical consequences that are picked up by MAST include only cirrhosis or an identification by physicians that alcohol is the etiology of medical complaints. However, previous studies have demonstrated low physician recognition of alcoholism<sup>17,18,20</sup>. Since the medical consequences of alcoholism and sleep apnea (sleep disturbance, depressive symptoms, hypertension, arrhythmias, etc) are almost identical, symptoms may not have been attributed to the presence of AUD.

Our study also found that 40% of MAST positive patients did not report hazardous consumption. This discrepancy is most likely related to the sleep laboratory policy of advising SAS patients to discontinue drinking. Patients referred to the sleep laboratory are generally quite symptomatic with their sleep disturbance and are routinely informed that alcohol can worsen their symptoms. In addition, physicians who refer patients for sleep studies commonly give the patient the same caveat because of the association between alcohol ingestion and apneic events. Thus, at the time the alcohol consumption portion of the sleep questionnaire was completed, it was unlikely that patients would report hazardous consumption. Lee and DeFrank point out that MAST may not be an accurate reflection of current alcohol consumption and may misidentify a person who no longer consumes alcohol<sup>21</sup>. However, since our study evaluates the possible toxic effect of chronic alcohol consumption, MAST is a useful tool to identify subjects who may have had a past history of hazardous drinking.

To get a more accurate indication of the prevalence of AUD in SAS patients, it is necessary to add the number of MAST positive patients to the number of MAST negative patients who indicated hazardous consumption. This yields a combined prevalence of 31%, 38% in males alone. The high prevalence in males is important because both AUD and sleep apnea are predominantly male diseases.

The 31% combined prevalence of AUD in SAS patients found in this study suggests an association between AUD and sleep apnea. This association cannot be explained on the basis of acute ingestion of alcohol because patients at the sleep laboratory who ingested alcohol prior to the visit were generally not evaluated by polysomnography. In addition, sleep apnea patients with AUD were more likely to have an AI of > 20, indicating there may be an association between AUD and the

severity of SAS.

In our study, a sub-sample of 17 of the 68 SAS identified 8 with AUD. None of the 8 had been referred by their physicians for alcohol counseling, although 4 had discontinued or cut down their drinking. As in other studies demonstrating low rates of recognition by physicians and referral for alcoholism treatment<sup>17,18,20</sup>, the concern is that complete evaluation of SAS patients should include screening and referral for AUD. This is because acute alcohol ingestion aggravates SAS and patients with AUD are unlikely to quit drinking for prolonged periods without the help of a recovery program. The results of our study suggest that patients with AUD are more likely to present with an AI of 20 or more; He et al demonstrated this degree of severity carries a significant risk for mortality<sup>8</sup>. Therefore, intervention in AUD is as important as therapy in sleep apnea.

The 31% combined prevalence of AUD in SAS patients and the likelihood of having an AI of 20 or more, suggests that the toxicity of chronic alcohol ingestion plays a role in the development of morbidity and mortality in sleep apnea. As a corollary, the morbidity associated with alcoholism may be attributed in part to the medical sequelae of sleep apnea (arrhythmias, hypertension, etc), especially since these are frequently found in alcoholic patients. This raises the question whether sleep apnea may be a common but undiagnosed problem in chronic alcoholics. The finding by Vitiello and colleagues of nocturnal hypoxia in abstaining alcoholics suggests this to be the case<sup>9,10</sup>. Further studies are needed in order to evaluate the association between alcoholism and sleep apnea.

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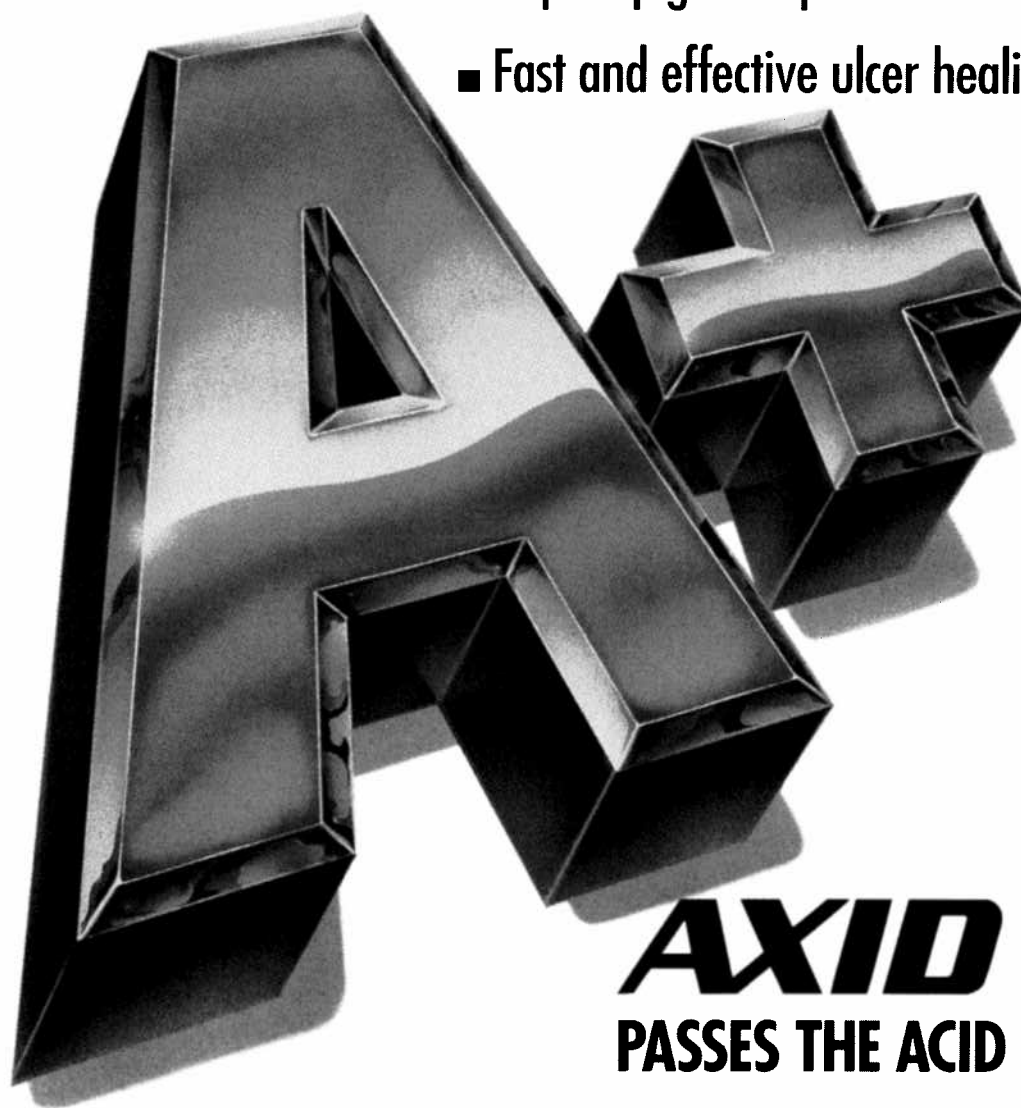
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**Carcinogenesis, Mutagenesis, Impairment of Fertility:** A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

**Pregnancy—Teratogenic Effects—Pregnancy Category C**—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

**Nursing Mothers:** Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

**Pediatric Use:** Safety and effectiveness in children have not been established.

**Use in Elderly Patients:** Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

**Adverse Reactions:** Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events were due to the drug.

**Hepatic:** Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

**Cardiovascular:** In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

**CNS:** Rare cases of reversible mental confusion have been reported.

**Endocrine:** Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

**Hematologic:** Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H<sub>2</sub>-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

**Integumental:** Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

**Hypersensitivity:** As with other H<sub>2</sub>-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

**Other:** Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

**Overdosage:** Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis does not substantially increase clearance of nizatidine due to its large volume of distribution.

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## ALCOHOL (Continued from page 285)

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## RBRVS (Continued from page 279)

worse off. No one can say that change won't be coming. Those who don't like the RBRVS and the limits on balance billing should consider the alternatives: mandatory assignment, MD-DRGs, and fees set by the government without any professional input. The RBRVS gives the profession a voice, and it enables us to ward off more objectionable measures.

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That means opposing policies that will undermine the RBRVS (such as a behavioral assumption that would lower the fee schedule conversion factor). It means working to change policies — such as the ban on reimbursement for EKG interpretation — that give with one hand and take away with the other. And it means supporting further changes that will make the system even better.

The RBRVS unites physicians under one fair and rational payment system to fight future detrimental budget cuts in Medicare. Lawmakers faced with a divided house of medicine easily can use that division to cut Medicare payments even further. But if they're faced with a profession that's united under the RBRVS, it won't be easy.

Support for the RBRVS has been right — for our profession and for our patients. The RBRVS will protect undervalued evaluation and management services in an era of Medicare budget cutting, increase access and the emphasis on preventive care for patients, and introduce fairness into the Medicare payment system. *But we must fight together — as a profession — to make sure it is implemented in the way Congress intended.*